ABSTRACTS

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Statin therapy, LDL cholesterol, C-reactive protein, and coronary artery disease

Nissen SE, Tuzcu EN, Schoenhagen P, et al. N Engl J Med 2005;352:29-38.

Conclusion: The reduced rate of the progression of atherosclerosis with intensive statin therapy compared with moderate statin therapy in patients with coronary disease is related to reductions in levels of C-reactive protein (CRP) and atherogenic proteins.

Summary: The authors sought to determine whether evidence from recent trials that demonstrates better outcomes with intensive versus moderate statin therapy (N Engl J Med 2004;350:1495-504 and JAMA 2004; 291:1071-80) is related to reductions in levels of CRP as well as reductions in levels of atherogenic proteins. Patients were randomly allocated to moderate treatment with 40 mg of oral pravastatin per day or intensive treatment with 80 mg of atorvastatin per day. All patients had angiographically documented coronary disease and were followed with intravascular ultrasound initially and at 18 months to measure progression of atherosclerosis. CRP and lipoprotein levels were measured at baseline and follow-up.

The mean low-density lipoprotein (LDL) cholesterol level decreased from 150.2mg/dL at baseline to 94.5 mg/dL in the group as a whole at 18 months ($P < .001$). CRP levels also decreased from 2.9 to 2.3 mg/dL ($P < .001$). There was a weak correlation between the reduction in LDL cholesterol levels and CRP levels ($r = 0.13$, $P = .005$). This reduction held for the group as a whole but was not significant in either treatment group alone. With univariant analysis, percent changes in CRP, LDL cholesterol, apolipoprotein B-100, and nonhigh density lipoprotein cholesterol were related to rates of progression of atherosclerosis. Adjusting for the reduction in lipid levels, decreases in CRP were independently and significantly correlated with rates of progression of atherosclerosis. Those patients with reductions in both CRP and LDL cholesterol greater than the median had significantly slower rates of progression than patients with reductions of both CRP and LDL cholesterol that were less than the median ($P = .001$).

Comment: This is another bit of evidence that statin-mediated reductions in LDL cholesterol and CRP are, for the most part, unrelated. Reductions in levels of atherogenic proteins in this study were not closely correlated with reductions in CRP levels. This study, along with a similar study in the same issue of the *New England Journal of Medicine* (2005;352: 20-8), suggests monitoring of both CRP and atherogenic protein levels in patients on statin therapy.

Effectiveness and safety of dialysis vascular access procedures performed by interventional nephrologists

Beathard GA, Litchfield T, and Physician Operators Forum of RMS Lifeline, Inc. Kidney Int 2004:66:1622-32.

Conclusion: Appropriately trained interventional nephrologists can performed basic procedures essential to the maintenance of hemodialysis access.

Summary: An interventional nephrologist performs interventional procedures pertinent to management of patients with renal disease. These procedures have traditionally included renal biopsy and insertion of temporary dialysis venous catheters. Some interventional nephrologists are now performing dialysis vascular access maintenance procedures. These include tunneled catheter placement and exchange, angioplasty, and thrombolysis procedures on synthetic grafts and native fistulas. The authors analyzed data on six hemodialysis access maintenance procedures performed by interventional nephrologists. These included angioplasty of arteriovenous fistulas (AVF-PTA), angioplasties of synthetic grafts (graft-PTA), thrombectomy of arteriovenous fistulas (AVF declot), thrombectomy of synthetic grafts (Graft declot), placement of tunneled dialysis catheters (TDC), and placement and tunneled dialysis catheter exchange (TDC exchange). Data were derived from 11 free-standing outpatient facilities in the United States. All but one were private practice nephrology groups. A total of twenty-nine interventional nephrologists were involved in performance of the procedures.

A total of 14,067 cases were performed, and 96.18% were successful. The minor complication rate was 3.26% and primarily included minor bleeding and reactions to medications. The major complication rate was 0.82% and included significant hematomas, pneumothorax, peripheral artery embolism, and two deaths within 30 days of performance of a procedure. Cases performed in each category with success rates were as follows: TDC placement–1,765 cases, 98.24% successful; TDC exchange–2,262 cases, 98.36% successful; AVF-PTA-1-561 cases, 96.58% successful; graft-PTA–3,560 cases, 98.06% successful; AVF declot–228 cases, 78.10% successful; graft declot–4,671 cases, 93.08% successful.

Comment: These cases were performed in an angiography suite. None involved open surgical procedures with the creation of arteriovenous fistulas or placement of synthetic grafts or surgical revisions of fistulas or grafts. The report is very simplistic in that long-term results were not considered. No life-table analysis is presented. However, it does appear nonsurgically trained and nonradiologically trained physicians can learn catheter-based procedures pertinent to maintenance of hemodialysis access.

Accelerated enlargement of experimental abdominal aortic aneurysms in a mouse model of chronic cigarette smoke exposure.

Buckley C, Wyble CW, Borhani M, et al. J Am Coll Surg 2004;199896-903.

Conclusion: In a model of experimental abdominal aortic aneurysms (AAAs), short-term exposure to cigarette smoke did not effect development of the aneurysm. Chronic smoke exposure was associated with a significant increase in late aneurysm dilatation.

Summary: The biologic mechanisms linking cigarette smoke and emphysema with AAA are unknown. The study was designed to determine if cigarette smoking enhances the growth of an experimental AAA. Experimental AAAs were created with elastase perfusion of the abdominal aorta in 129/SdEb mice. The experimental mice were subject to chronic cigarette smoking for 2 weeks, followed by elastase perfusion of the abdominal aorta. Smoking was then continued for either 2 or 12 weeks. Twenty-nine nonsmoking control mice also under went elastase perfusion and follow-up evaluation at the same time intervals. Aortic diameters were measured to determine increases in aortic diameter (ΔAD). An abdominal aortic aneurysm was defined as a ΔAD >100%.

The experimental groups did not differ with regard to preperfusion and immediate postperfusion ADs. In both smoking and nonsmoking controls, aneurysm dilatation was present 2 weeks after elastase perfusion, with no significant difference in the final AD. AAAs developed in all animals by 12 weeks after elastase perfusion. Aorta dilatation was 50% greater in smoking mice compared with nonsmoking controls (ΔAD smoking, 204% ± 23% vs non smoking, 135% ± 17%; $P < .05$).

Comment: This study serves as a first step toward determining how cigarette smoking may influence the development of aneurysms. It represents a combination of two well-characterized experimental models, an elastase-induced model of AAA and a model of cigarette smoke-induced pulmonary emphysema. The study indicates it is feasible to combine the two models in a single animal. It may eventually yield clues as to the mechanism of tobacco-induced aortic degeneration.

Incidence of deep vein thrombosis after varicose vein surgery.

van Rij AM, Chai J, Hill GB, Christie RA. Br J Surg 2004;91:1582-1585.

Conclusions: A measurable incidence of deep vein thrombosis (DVT) occurs after varicose vein surgery. DVT after varicose vein surgery has minimal short- and long- term clinical significance.

Summary: This was a prospective study to examine the incidence of DVT in patients undergoing varicose vein surgery. Leg veins were examined before varicose vein surgery by duplex ultrasound scanning in 377 patients. The veins were reassessed at 2 to 4 weeks after surgery and again at 6 and 12 months. Patients were instructed to contact a physician if symptoms consistent with DVT occurred before any scheduled follow-up appointment. Subcutaneous heparin for DVT prophylaxis was used at the discretion of the operating surgeon.

DVT was detected in 20 (5.3%) of the 377 patients. Only eight DVT were symptomatic, and no patient developed symptoms consistent with pulmonary embolism. Of the 20 DVT detected, 18 were confined to calf veins. Subcutaneous heparin as a prophylactic agent prior to varicose vein surgery did not appear to alter outcome, with 64.7% of patients receiving prophylaxis, and 5.7% of patients who received prophylaxis developing DVT. No patient aged ≤40 years in this study developed a DVT with varicose vein surgery. Risk factors for DVT with varicose vein surgery were CEAP classes 5 and 6 and a positive family history of DVT. Obesity and oral contraceptives were not risk factors for DVT after varicose vein surgery. None of the thrombi found in this study progressed, and half resolved within 1 year, without evidence of venous reflux in the deep veins.

Comment: Prophylaxis for venous thrombosis in patients undergoing varicose vein surgery is controversial. Based on the results of this study, the current guidelines suggested by the American College of Chest Physicians and the Scottish Guideline Network to limit DVT prophylaxis in varicose vein surgery to those patients with risk factors for DVT appears appropriate. Patients with multiple risk factors perhaps should also be considered for more extended prophylaxis.